culture is only a sampling procedure, at best, and might miss meningococci even if present. Finally, one could argue with some reason that the person in whom a culture is negative may be at greater risk of disease than the person who is already carrying meningococci in an asymptomatic fashion, since the carrier state is generally an immunizing event.

Perhaps the greatest psychological pressures for chemoprophylaxis are brought to bear in the case of nonhousehold contacts, particularly classroom contacts. Utilizing the opportunity provided by the recent epidemic in Brazil, Jacobson and his colleagues[7] convincingly showed that there was no significantly increased risk associated with classroom exposure to a student with meningococcal meningitis; thus, no recommendation for chemoprophylaxis of classroom contact can be made.

Science ends at this point with regard to chemoprophylaxis, and the art of medicine must take over. Recognizing the almost hysterical fear that sometimes occurs in persons exposed to this disease, the understanding and perceptive physician may sometimes elect, quite justifiably, to provide chemoprophylaxis for psychological reasons alone.

The companion report in this issue by Oill and her associates provides a vivid illustration of a "household" outbreak and illustrates what can happen when an unusually invasive strain is disseminated into a susceptible population. Such dramatic outbreaks are fortunately infrequent, but invariably one wonders just what it was about that particular strain of group B Neisseria meningitidis that made it so highly invasive? Are there detectable differences between strains with obvious disease producing potential and the other strains that so many of us carry around harmlessly (perhaps beneficially) in our nasopharynges? Answers to this question are slowly emerging, and appear to be distinctly affirmative. Both Frasch and Chapman[8] and Gold and co-workers[9] have been investigating antigens of Neisseria meningitidis other than the serogroup (A, B, C, and so forth) capsular antigens. Subcapsular protein antigens, localized to the outer membrane of the cell envelope, have been identified, at least some of which are common to all the usual serogroups of meningococci.

Work recently reported by Griffiss and associates from Walter Reed Army Institute of Research[10] suggests that one or more of such subcapsular antigens may be associated with invasiveness or epidemic potential. Therefore, certain antigens are almost invariably present in meningococci isolated from cases, but generally absent in organisms isolated from healthy carriers. These findings raise the possibility that protein subcapsular antigens may ultimately form the basis for another approach to immunization against meningococcal disease. Although polysaccharide vaccines against both group A and group C meningococci have been highly effective, no satisfactory polysaccharide vaccine directed against group B meningococci has been developed. If antibody directed against protein subcapsular antigens can be shown to be protective, it may prove possible to prepare vaccines containing such protein antigens that will, in effect, protect against meningococci of all serogroups. Such an approach seems well worth pursuing.

THEODORE C. EICKHOFF, MD
Professor of Medicine
Head, Division of Infectious Disease
University of Colorado Medical Center
Denver

REFERENCES

1. Summary: Cases of specified notifiable diseases—United States. Atlanta, Center for Disease Control, Morbidity and Mortality Weekly Report 27:2, Jan 6, 1978
2. McCormick JB, Bennett JV: Public health considerations in the management of meningococcal disease. Ann Intern Med 83:883-886, Dec 1975
3. Artenstein MS: Prophylaxis for meningococcal disease. JAMA 231:1035-1037, Mar 10, 1975
4. The Meningococcal Disease Surveillance Group: Meningococcal disease—Secondary attack rate and chemoprophylaxis in the United States, 1974. JAMA 235:261-265, Jan 19, 1976
5. Khouri-Boulos N: Meningococcal meningitis following rifampin prophylaxis. Am J Dis Child 126:689-691, 1973
6. Munford RS, Taunay AE, De Morais JS, et al: Spread of meningococcal infection within households. Lancet 1:1275-1278, Jun 1974
7. Jacobson JA, Camargos PAM, Ferreira JT, et al: The risk of meningitis among classroom contacts during an epidemic of meningococcal disease. Am J Epidemiol 104:552-555, 1976
8. Frasch CE, Chapman SS: Classification of Neisseria meningitidis group B into distinct serotypes—III. Application of a new bactericidal inhibition technique to distribution of serotypes among cases and carriers. J Infect Dis 127:149-154, Feb 1973
9. Gold R, Winglehake JL, Mars RS, et al: Identification of an epidemic strain of group C Neisseria meningitidis by bactericidal serotyping. J Infect Dis 124:593-597, Dec 1971
10. Griffiss JM, Broud DD, Silver CA, et al: Immuno-epidemiology of meningococcal disease in military recruits—I. A model for serogroup independency of epidemic potential as determined by serotyping. J Infect Dis 136:176-186, 1977

Public Views of NHI

DURING THE MONTH of October 1977 the Department of Health, Education, and Welfare conducted an intensive outreach effort to solicit the public's views of national health insurance (NHI). Public hearings were held in every state and more than 8,600 persons and organizations provided oral or written comments. Health professionals and professional organizations and other medical groups were well represented, as were the elderly, the general public, insurance companies and many others. A report of this considerable effort has recently become available.[1]

A genuine attempt seems to have been made to involve all who should or might want to be involved. The result was something like a national town meeting with all the various interests presenting their views on more or less equal footing. The wide spectrum of opinions, some informed and some probably not so well informed, is candidly revealed. Few if any clear directions or recommendations emerged, and it is to be hoped that our political and bureaucratic leadership takes heed and proceeds cautiously.

In a way this approach seems like a breath of fresh air. Those most concerned and interested in this complex problem were being asked rather than being told by government, and the response has the ring of truth. It is possible that a first step has been taken toward solving a major social problem by seeking something approaching a national consensus as opposed to arbitrary legislative fiat or perhaps an imposition of the will of a determined minority. In this exercise one can see the germ of a new more democratic and more participatory approach to solving some of the more difficult social and economic problems of modern America, an approach that will involve all those who should probably be involved in decision making and one that is in the tradition of true participatory democracy which is one of our nation's great heritages. It is to be hoped that the proponents of this approach will not be discouraged by the lack of clear direction or recommendation for NHI at this time. This is simply the way it is. But this outreach effort can be viewed optimistically as an essential first move toward a consensus that will truly reflect the will of the people on this important matter. —MSMW

REFERENCE

1. National Health Insurance National Outreach Report. U.S. Department of Health, Education, and Welfare, Dec 1977. U.S. Government Printing Office 1978—793—675/158.

Patent Ductus Arteriosus

THAT A PERSISTENTLY patent ductus arteriosus can produce problems in preterm infants is one of the few clearly established facts in the field. Although complete data are not yet available, the incidence of prematurity is about 15 per 1,000 live births and of these infants about half (8 per 1,000 live births) have a patent ductus arteriosus. The recognition, significance and management of a patent ductus arteriosus are still topics of vigorous debate among neonatologists and pediatric cardiologists. The Specialty Conference in this issue has presented some of the specific views of the group in San Diego. These views, and some of the concepts presented, do not necessarily agree with those of others working in the same field; this points out the lack of agreement and standardization that exists. These authors have touched on a few of the questions that are currently posed, but unfortunately have not been able to come up with any new clear answers or unifying recommendations. They also have not touched on several very important topics. This failure is not completely for lack of attempt at illuminating the problem, but rather reflects our sadly incomplete understanding of the physiology and pathophysiology of the ductus arteriosus.

It is unfortunate that Dr. Gluck feels that pediatric cardiologists are not an integral part of the overall care of small preterm infants in the intensive care nurseries but rather should be invited only in a consultant role. This is understood when one considers the high incidence of patent ductus arteriosus and that the major advances in diagnosis and management of infants with this disorder come from cardiologists. Even more in keeping with a tandem approach to management of all preterm infants is the not infrequent occurrence of the very subtle signs of a patent ductus arteriosus (and occasionally no signs) as described. These may well require the early use of noninvasive techniques, such as echocardiography, as well as possible invasive techniques, such as retrograde aortography. These both require the services of pediatric cardiologists.

Although important, recognition of the presence of a patent ductus arteriosus with either obvious or subtle signs is not as critical as recognition of the significance of the hemodynamic alterations produced by the left-to-right shunt through the patent ductus arteriosus. Duration and volume of murmurs are notoriously misleading, pulse volume and pulse pressure are frequently increased in normal premature infants, and many of the clinical signs typical of patent ductus arteriosus in older children are not present in premature infants. Echocardiographic assessment of left atrial size as a reflection of shunt magnitude has been a valuable addition to management. As with all individual components of diagnosis, however, an increase in left atrial size can be easily misread or misinterpreted. Changes in blood volume without a shunt could well affect left atrial size. Likewise, variations in ventilator pressures affect the measured left atrial size. An increased left atrial dimension, therefore, does not always mean a patent